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Department of Neurosciences, University of California, S Diego, School of Medicine La Jolla, 92093-0624, USA.

The precursor of the non-Abeta component of Alzheimer' disease amyloid (NACP) (also known as a-synuclein) is a presynaptic terminal molecule that accumulates in the pla of Alzheimer's disease. Recent studies have shown that a mutation in NACP is associated with familial Parkinson's disease, and that Lewy bodies are immunoreactive with antibodies against this molecule. To clarify the patterns o accumulation and differences in abnormal compartmentalization, we studied NACP immunoreactivi using double immunolabeling and laser scanning confoca microscopy in the cortex of patients with various neurodegenerative disorders. In Lewy body variant of Alzheimer's disease, diffuse Lewy body disease, and Parkinson's disease, NACP was found to immunolabel cortical Lewy bodies, abnormal neurites, and dystrophic neurites in the plaques. Double-labeling studies showed tl all three of these neuropathological structures also contair

ubiquitin, synaptophysin, and neurofilament (but not tau) immunoreactivity. In contrast, neurofibrillary tangles, neuropil threads, Pick bodies, ballooned neurons, and glia tangles (most of which were tau positive) were NACP negative. These results support the view that NACP specifically accumulates in diseases related to Lewy bodi such as Lewy body variant of Alzheimer's disease, diffuse Lewy body disease, and Parkinson's disease and suggests role for this synaptic protein in the pathogenesis of neurodegeneration.

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Abnormal distribution of the non-Abeta compor of Alzheimer's disease amyloid precursor/alpha-synuclein in Lewy body disease as revealed by proteinase K and formic acid pretreatment.

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The precursor of the non-Abeta component of Alzheimer' disease amyloid (NACP) (also known as alpha-synuclein) presynaptic terminal molecule that abnormally accumulat the plaques of Alzheimer's disease (AD) and in the Lewy bodies (LBs) of Lewy body variant of AD, diffuse Lewy body disease, and Parkinson's disease. To better understar the distribution of NACP/alpha-synuclein and its fragmen the LB-bearing neurons and neurites, as well as to clarify patterns of NACP/alpha-synuclein compartmentalization, studied NACP/alpha-synuclein immunoreactivity using antibodies against the C-terminal, N-terminal, and NAC regions after Proteinase K and formic acid treatment in th cortex of patients with LBs. Furthermore, studies of the subcellular localization of NACP/alpha-synuclein within bearing neurons were performed by immunogold electron

microscopy. These studies showed that the N-terminal antibody immunolabeled the LBs and dystrophic neurites with great intensity and, to a lesser extent, the synapses. I contrast, the C-terminal antibody strongly labeled the synapses and, to a lesser extent, the LBs and dystrophic neurites. Whereas Proteinase K treatment enhanced NACP/alpha-synuclein immunoreactivity with the C-term antibody, it diminished the N-terminal NACP/alpha-synu immunoreactivity. Furthermore, formic acid enhanced LB and dystrophic neurite labeling with both the C- and N-terminal antibodies. In addition, whereas without pretreatment only slight anti-NAC immunoreactivity was found in the LBs, formic acid pretreatment revealed an extensive anti-NAC immunostaining of LBs, plaques, and glial cells. Ultrastructural analysis revealed that NACP/al synuclein immunoreactivity was diffusely distributed wit the amorphous electrodense material in the LBs and as sm clusters in the filaments of LBs and neurites. These result support the view that aggregated NACP/alpha-synuclein might play an important role in the pathogenesis of disord associated with LBs.

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Immunohistochemical localization of amyloid be protein with amino-terminal aspartate in the cerebral cortex of patients with Alzheimer's disease.

Arai T, Akiyama H, Ikeda K, Kondo H, Mori H.

Department of Neuropathology, Tokyo Institute of Psychiatry, 2-1-8 Kamikitazawa, Setagaya-ku, Tokyo 156 8585, Japan. arai@prit.go.jp

We investigated immunohistochemically the localization amyloid beta-protein (Abeta) with amino-terminal asparta (N1[D]) in brains of patients with Alzheimer's disease, diffuse Lewy body disease and Down's syndrome. A monoclonal antibody, 4G8, which recognizes the middle portion of Abeta, was used as a reference antibody to labe the total Abeta deposits. Double staining with anti-Abeta([D]) and 4G8 revealed that Abeta deposits in the subiculu and the neocortical deep layers often lacked N1[D] immunoreactivity, indicating N-terminal truncation of Ab in these deposits. Abeta deposits in the neocortical superf layers and the presubicular parvopyramidal layer always contained Abeta with N1[D]. Such regional as well as lam differences in the distribution of Abeta beginning at N1[D suggest that some local factors influence N-terminal

processing of Abeta deposited in the brain.

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Alpha-synuclein accumulates in Lewy bodies in Parkinson's disease and dementia with Lewy bo but not in Alzheimer's disease beta-amyloid plac cores.

Bayer TA, Jakala P, Hartmann T, Havas L, McLean C Culvenor JG, Li QX, Masters CL, Falkai P, Beyreuth K.

Department of Psychiatry, University of Bonn Medical Center, Germany. bayer@uni-bonn.de

A growing body of evidence suggests that the non-Abeta component of Alzheimer's disease amyloid precursor prot (NACP) or alpha-synuclein contributes to the neurodegenerative processes in Alzheimer's disease (AD) Parkinson's disease (PD) and dementia with Lewy bodies (DLB). In the present study antisera to the N terminus anc NAC domain of the alpha-synuclein protein were employ to elucidate the expression pattern in brains of patients wi AD, PD, DLB and control specimen. Alpha-synuclein exhibited an overall punctuate expression profile compati with a synaptic function. Interestingly, while Lewy bodie were strongly immunoreactive, none of the alpha-synucle antisera revealed staining in mature beta-amyloid plaques AD. These observations suggest that alpha-synuclein doe

contribute to late neurodegenerative processes in AD brai

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Deposition of beta-amyloid subtypes 40 and 42 differentiates dementia with Lewy bodies from Alzheimer disease.

Lippa CF, Ozawa K, Mann DM, Ishii K, Smith TW, Arawaka S, Mori H.

Department of Neurology, MCP-Hahnemann University, Philadelphia, PA 19129, USA. lippa@auhs.edu

BACKGROUND: Alterations in the metabolism of the amyloid precursor protein and the formation of beta-amyl (Abeta) plaques are associated with neuronal death in Alzheimer disease (AD). The plaque subtype Abeta(x-42) occurs as an early event, with Abeta(x-40) plaques formir a later stage. In dementia with Lewy bodies (DLB), an increase in the amount of cortical Abeta occurs without severe cortical neuronal losses. OBJECTIVE: To advance understanding of the natural history of Abeta in neurodegenerative diseases. DESIGN: We evaluated the expression of Abeta(x-40) and Abeta(x-42) in DLB using monoclonal antibodies and immunohistochemical techniq in 5 brain regions. The data were compared with those elicited with normal aging and from patients with AD. SETTING AND PATIENTS: A postmortem study involv 19 patients with DLB without concurrent neuritic

degeneration, 10 patients with AD, and 17 aged persons without dementia for control subjects. RESULTS: The Al plaques were more numerous in patients with DLB than ii controls in most brain regions, although the Abeta(x-42) plaque subtype was predominant in both conditions. Over Abeta(x-42) plaque density was similar in patients with D and those with AD, but Abeta(x-40) plaques were more numerous in persons with AD than in those with DLB. Tl ratio of Abeta(x-40) to Abeta(x-42) plaques was significa reduced in persons with DLB compared with patients witl AD. CONCLUSIONS: The Abeta plaques were more numerous in patients with DLB than persons with normal aging, but the plaque subtypes were similar. The relative proportion of the 2 Abeta plaque subtypes in DLB is distinguishable from that in AD.

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ARCH NEUROL

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Non-Abeta component of Alzheimer's disease amyloid (NAC) revisited. NAC and alpha-synucl are not associated with Abeta amyloid.

Culvenor JG, McLean CA, Cutt S, Campbell BC, Mal F, Jakala P, Hartmann T, Beyreuther K, Masters CL, QX.

Department of Pathology, The University of Melbourne, Parkville, Victoria, Australia.

alpha-Synuclein (alphaSN), also termed the precursor of t non-Abeta component of Alzheimer's disease (AD) amylc (NACP), is a major component of Lewy bodies and Lewy neurites pathognomonic of Parkinson's disease (PD) and dementia with Lewy bodies (DLB). A fragment of alphaS termed the non-Abeta component of AD amyloid (NAC) previously been identified as a constituent of AD amyloid plaques. To clarify the relationship of NAC and alphaSN Abeta plaques, antibodies were raised to three domains of alphaSN. All antibodies produced punctate labeling of hu cortex and strong labeling of Lewy bodies. Using antibod

to alphaSN(75-91) to label cortical and hippocampal secti of pathologically proven AD cases, we found no evidence NAC in Abeta amyloid plaques. Double labeling of tissue sections in mixed DLB/AD cases revealed alphaSN in dystrophic neuritic processes, some of which were in clos association with Abeta plaques restricted to the CA1 hippocampal region. In brain homogenates alphaSN was predominantly recovered in the cytosolic fraction as a 16- protein on Western analysis; however, significant amount aggregated and alphaSN fragments were also found in ure extracts of SDS-insoluble material from DLB and PD cas NAC antibodies identified an endogenous fragment of 6 k the cytosolic and urea-soluble brain fractions. This fragm may be produced as a consequence of alphaSN aggregatic alternatively may accelerate aggregation of the full-length alphaSN.

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Cellular co-localization of phosphorylated tau- a NACP/alpha-synuclein-epitopes in lewy bodies i sporadic Parkinson's disease and in dementia wi Lewy bodies.

Arima K, Hirai S, Sunohara N, Aoto K, Izumiyama Y, Ueda K, Ikeda K, Kawai M.

Department of Ultrastructure and Histochemistry, Tokyo Institute of Psychiatry, 2-1-8 Kamikitazawa, Setagaya-ku, Tokyo, Japan. arima@prit.go.jp

The precursor of the non-Abeta-component of Alzheimer' disease (AD) amyloid (NACP, alpha-synuclein) aggregat into insoluble filaments of Lewy bodies (LBs) in Parkinsc disease (PD) and dementia with LBs (DLB). The microtubule-associated protein tau is an integral compone of filaments of neurofibrillary tangles (NFTs). NFTs are occasionally found in brains of PD and DLB; however, th presence of NFTs or tau-epitopes within LB-containing neurons is rare. Double-immunofluorescence study and peroxidase-immunohistochemical study in serial sections, performed to examine the co-localization of tau- and NAC epitopes in the brainstem of PD and DLB, demonstrated t four different epitopes of tau including phosphorylation-dependent and independent ones were present in a minori

LBs, but more often than previously considered. A tau (ta
epitope was localized to filaments in the outer layers of
brainstem-type LBs by immunoelectron microscopy.
Therefore, we conclude that tau is incorporated into filam
in certain LBs. Extensive investigation has enabled us to
classify this co-localization into four types: type 1, LBs w
ring-shaped tau-immunoreactivity; type 2, LBs surrounde
NFTs; type 3, NACP- and tau-immunoreactive filamentou
and granular masses; and type 4, NACP- and tau-
immunoreactive dystrophic neurites. This study raises a n
question whether aggregation and hyperphosphorylation c
tau in PD and DLB are triggered by the collapse of
intraneuronal organization of microtubules due to NACP-
filament aggregation in neuronal perikarya and axons.

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Cellular co-localization of phosphorylated tau- a NACP/alpha-synuclein-epitopes in lewy bodies i sporadic Parkinson's disease and in dementia wi Lewy bodies.

Arima K, Hirai S, Sunohara N, Aoto K, Izumiyama Y, Ueda K, Ikeda K, Kawai M.

Department of Ultrastructure and Histochemistry, Tokyo Institute of Psychiatry, 2-1-8 Kamikitazawa, Setagaya-ku, Tokyo, Japan. arima@prit.go.jp

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Widespread occurrence of alpha-synuclein/NAC immunoreactive neuronal inclusions in juvenile adult-onset Hallervorden-Spatz disease with Lew bodies.

Wakabayashi K, Yoshimoto M, Fukushima T, Koide H Horikawa Y, Morita T, Takahashi H.

Brain Disease Research Center, Brain Research Institute, Niigata University, Japan.

Alpha-Synuclein (originally called precursor of the non-Abeta component of Alzheimer's disease amyloid-NACP) presynaptic nerve terminal protein and is now known to b major component of Lewy bodies (LBs) in Parkinson's disease. Previous studies have shown that LBs are occasionally found in patients with Hallervorden-Spatz disease (HSD), a hereditary or sporadic neuroaxonal dystrophy. Therefore, an immunocytochemical examinati of the brain tissues from two patients with HSD for alpha-synuclein/NACP was performed. In both cases, LBs were observed in the substantia nigra, locus ceruleus and other subcortical nuclei. These LBs were strongly immunolabel with anti-alpha-synuclein/NACP. Moreover, abnormal al

synuclein/NACP-immunoreactive structures in the neuror
somata and processes were found in the cerebral neocorte
hippocampus, basal ganglia, thalamus, pontine and inferic
olivary nuclei, spinal grey matter, and peripheral sympath
ganglia. Although numerous dystrophic axons (spheroids)
were found throughout the brain, either none or only a fev
were positive for alpha-synuclein/NACP. These findings
suggest that widespread accumulation of alpha-synuclein/NACP is a pathological feature in patients suffe
from HSD with LBs, and that this phenomenon is unrelate
axonal spheroid formation.

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ACS PUBLICATIONS

Fibrils formed in vitro from alpha-synuclein and two mutant forms linked to Parkinson's disease typical amyloid.

Conway KA, Harper JD, Lansbury PT Jr.

Center for Neurologic Diseases, Brigham and Women's Hospital and Department of Neurology, Harvard Medical School, Boston, Massachusetts 02115, USA.

Two missense mutations in the gene encoding alpha-synuclein have been linked to rare, early-onset forms of Parkinson's disease (PD). These forms of PD, as well as tl common idiopathic form, are characterized by the presen cytoplasmic neuronal deposits, called Lewy bodies, in the affected region of the brain. Lewy bodies contain alpha-synuclein in a form that resembles fibrillar Abeta derived from Alzheimer's disease (AD) amyloid plaques. One of t mutant forms of alpha-synuclein (A53T) fibrillizes more rapidly in vitro than does the wild-type protein, suggestin that a correlation may exist between the rate of in vitro fibrillization and/or oligomerization and the progression c PD, analogous to the relationship between Abeta fibrilliza in vitro and familial AD. In this paper, fibrils generated ir

vitro from alpha-synuclein, wild-type and both mutant for are shown to possess very similar features that are characteristic of amyloid fibrils, including a wound and predominantly unbranched morphology (demonstrated by atomic force and electron microscopies), distinctive dye-binding properties (Congo red and thioflavin T), and antiparallel beta-sheet structure (Fourier transform infrare spectroscopy and circular dichroism spectroscopy). alpha-Synuclein fibrils are relatively resistant to proteolysis, a property shared by fibrillar Abeta and the disease-associa fibrillar form of the prion protein. These data suggest that like AD, is a brain amyloid disease that, unlike AD, is characterized by cytoplasmic amyloid (Lewy bodies). In addition to amyloid fibrils, a small oligomeric form of alp synuclein, which may be analogous to the Abeta protofib was observed prior to the appearance of fibrils. This speci or a related one, rather than the fibril itself, may be responsible for neuronal death.

PMID: 10704204 [PubMed - indexed for MEDLINE]

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Accumulation of insoluble alpha-synuclein in dementia with Lewy bodies.

Campbell BC, Li QX, Culvenor JG, Jakala P, Cappai Beyreuther K, Masters CL, McLean CA.

Department of Pathology, The University of Melbourne, 3010, Australia.

The alpha-synuclein (alpha SN) protein is thought to play central role in the pathogenesis of neurodegenerative dise where it aggregates to form intracellular inclusions. We h used Western blotting to examine the expression levels ar solubility of alpha SN in brain homogenates from dement with Lewy bodies (DLB), Parkinson's disease (PD), Alzheimer's disease (AD), and normal controls using sam from the parahippocampus/transentorhinal cortex. Compa to controls, DLB brains accumulate significantly greater amounts of sodium dodecyl sulfate (SDS)-soluble and SD insoluble alpha SN but levels of TBS-soluble alpha SN di not change. Levels of synaptophysin, a marker of synapti integrity, were significantly lower in DLB cases than in normal aged controls regardless of whether concurrent changes of AD were present. This limbic synaptic dysfunction may contribute to cognitive impairment in DI Whether aggregated alpha SN is a cause or effect of the

disease process in DLB and PD remains to be determined the presence of aggregated alpha SN is consistent with a pathogenesis similar to that associated with aggregates of Abeta amyloid in AD.

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Neuropathology of synuclein aggregates.

Duda JE, Lee VM, Trojanowski JQ.

Center For Neurodegenerative Disease Research, Univers
of Pennsylvania School of Medicine, Philadelphia, PA 19
USA.

Beginning with the isolation of the fragment of alpha-synuclein (alpha-syn) known as the non-Abeta componen amyloid plaques (NAC peptide) from Alzheimer's disease (AD) brains, alpha-syn has been increasingly implicated i the pathogenesis of neurodegenerative diseases, which no are classified as synucleinopathies. Indeed, unequivocal evidence linking abnormal alpha-syn to mechanisms of br degeneration came from discoveries of missense mutation the alpha-syn gene pathogenic for familial Parkinson's dis (PD) in rare kindreds. Shortly thereafter, alpha-syn was shown to be a major component of Lewy bodies (LBs) an Lewy neurites in sporadic PD, dementia with LBs (DLB) the LB variant of AD. Also, studies of brains from patient with AD caused by genetic abnormalities demonstrated m alpha-syn positive LBs. Further, alpha-syn was implicatec the formation of the glial (GCIs) and neuronal cytoplasmi inclusions of multiple system atrophy, and the LBs, GCIs neuraxonal spheroids of neurodegeneration with brain iro

accumulation type 1. Recently, two other members of the synuclein family, beta- and gamma-synuclein, have also t recognized to play a role in the pathogenesis of novel axo lesions in PD and DLB. Evidence for a role of alpha-syn i the formation of filamentous aggregates was reinforced b vitro studies showing aggregation and fibrillogenesis of mutant and wild type alpha-syn. Indeed, since the aggreg of brain proteins into presumptively toxic lesions is emer as a common but poorly understood mechanistic theme in sporadic and hereditary neurodegenerative diseases, clarification of the mechanism of synuclein aggregation c augment efforts to develop novel and more effective thera for many neurodegenerative disorders.

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Lewy body pathology in Alzheimer's disease.

Kotzbauer PT, Trojanowsk JQ, Lee VM.

Center for Neurodegenerative Disease Research, Universi of Pennsylvania School of Medicine, Philadelphia, USA.

Lewy bodies, the characteristic pathological lesion of substantia nigra neurons in Parkinson's disease (PD), are frequently observed to accompany the amyloid plaque an neurofibrillary tangle pathology of Alzheimer's disease (A However the typical anatomic distribution of Lewy bodie AD is distinct from PD. The most common site of occurre is the amygdala, where Lewy bodies are observed in approximately 60% of both sporadic and familial AD. Otl common sites of occurrence include the periamygdaloid a entorhinal cortex, while neocortical and brainstem areas develop Lewy bodies in a lower percentage of cases. In contrast, dementia with Lewy bodies (DLB), defined by widespread neocortical and brainstem Lewy bodies but frequently accompanied by variable levels of AD-type pathology, represents the other end of a spectrum of pathology associated with dementia. The observation of L bodies in familial AD cases suggests that like neurofibrill tangles, the formation of Lewy bodies can be induced by t pathological state caused by Abeta-amyloid overproducti

The role of Lewy body formation in the dysfunction and degeneration of neurons remains unclear. The protein alpl synuclein appears to be an important structural componen Lewy bodies, an observation spurred by the discovery of point mutations in the alpha-synuclein gene linked to rare cases of autosomal dominant PD. Further investigation of alpha-synuclein and its relationship to pathological condit promoting Lewy body formation in AD, PD, and DLB mε yield further insight into pathogenesis of these diseases.

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Biochem J

Binding of Abeta to alpha- and beta-synucleins: identification of segments in alpha-synuclein/NA precursor that bind Abeta and NAC.

Jensen PH, Hojrup P, Hager H, Nielsen MS, Jacobsen Olesen OF, Gliemann J, Jakes R.

Department of Medical Biochemistry, University of Aarh Ole Worms Alle, Building 170, DK-8000 Aarhus C, Denmark.

NAC, a 35-residue peptide derived from the neuronal prot alpha-synuclein/NAC precursor, is tightly associated with Abeta fibrils in Alzheimer's disease amyloid, and alpha-synuclein has recently been shown to bind Abeta in vitro. have studied the interaction between Abeta and synuclein aiming at determining segments in alpha-synuclein that ca account for the binding, as well as identifying a possible interaction between Abeta and the beta-type synuclein. W report that Abeta binds to native and recombinant alpha-synuclein, and to beta-synuclein in an SDS-sensitive interaction (IC50 approx. 20 microM), as determined by chemical cross-linking and solid-phase binding assays. al Synuclein and beta-synuclein were found to stimulate Ab aggregation in vitro to the same extent. The synucleins als

displayed Abeta-inhibitable binding of NAC and they we
capable of forming dimers. Using proteolytic fragmentati
of alpha-synuclein and cross-linking to 125I-Abeta, we
identified two consecutive binding domains (residues 1-5
and 57-97) by Edman degradation and mass spectrometri
analysis, and a synthetic peptide comprising residues 32-
possessed Abeta-binding activity. To test further the possi
significance in pathology, alpha-synuclein was biotinylat
and shown to bind specifically to amyloid plaques in a br
with Alzheimer's disease. It is proposed that the multiple
Abeta-binding sites in alpha-synuclein are involved in the
development of amyloid plaques.

PMID: 9163350 [PubMed - indexed for MEDLINE]

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Bodles AM, Guthrie DJ, Harriott P, Campbell P, Irvi GB.

Centre for Peptide and Protein Engineering, School of Biology and Biochemistry, The Queen's University of Belfast, Northern Ireland.

The non-Abeta component of Alzheimer's disease amyloi (NAC) and its precursor alpha-synuclein have been linke amyloidogenesis in Alzheimer's disease (AD), Parkinson' disease (PD) and dementia with Lewy bodies (DLB). Previously we have shown that NAC forms beta-sheet structures and fibrils [El-Agnaf, O.M.A., Bodles, A.M., Guthrie, D.J.S., Harriott, P. & Irvine, G.B. (1998) Eur. J. Biochem. 258, 157-163]. As a measure of their neurotoxi potential we have examined the ability of fresh and aged NAC and fragments thereof to inhibit the reduction of the redox dye 3-(4, 5-dimethylthiazol-2-yl)-2,5 diphenyltetrazolium bromide by rat pheochromocytoma P cells. Micromolar concentrations of NAC and fragments

thereof display varying degrees of toxicity. On immediate dissolution and after an incubation period for 3 days at 37 degrees C the full-length peptide and fragments NAC(3-1 and NAC(1-18) scrambled sequence [NAC(1-18 s)] were toxic, whereas fragments NAC(1-13) and NAC(6-14) wer not. CD indicates that NAC(3-18) and NAC(1-18 s) exhit beta-sheet secondary structure in aqueous solution, where NAC(1-13) and NAC(6-14) do not. NAC(3-18) aggregate as indicated by concentration of peptide remaining in solu after 3 days measured by an HPLC assay, and forms fibril as determined by electron microscopy. However, althougl some fibrils were detected for NAC(1-18 s) it does not co out of solution to a significant degree. Fragments NAC(1- and NAC(6-14) form few fibrils and remain in solution. These findings indicate that the ability of the central regio NAC to form beta-sheet secondary structures is important determining the toxicity of the peptide. This contrasts witl what has been reported previously for most Abeta peptide their toxicity appears to require the peptide to have forme fibrillary aggregates as well as displaying beta-sheet. The: results suggest that an intermediate, which exhibits beta-s structure, may be responsible for the toxic properties of N and provides further evidence for the role of NAC in the pathogenesis of AD, PD and DLB.

PMID: 10759841 [PubMed - indexed for MEDLINE]

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Alpha-synuclein aggregation and neurodegenerative diseases.

Ma QL, Chan P, Yoshii M, Ueda K.

Department of Neural Plasticity, Tokyo Institute of Psychiatry, Tokyo 156-8585, Japan.

Alpha-synuclein is a neuronal protein originally identifiec Alzheimer's disease (AD) amyloid plaques in 1993 and named non-Abeta component precursor (NACP) [92]. Lat the discovery of two missense mutations (G88C and G20 which resulted in Ala30Pro (A30P) and Ala53Thr (A53T substitutions, of the alpha-synuclein gene in certain autosomal-dominant early onset familial Parkinson's dise (PD) has greatly promoted the understanding of the role o alpha-synuclein in the pathogenesis of neurodegenerative diseases, such as PD, dementia with Lewy bodies (DLB) multiple system atrophy (MSA) [5,6,51,75]. At present, it widely accepted that alpha-synuclein may play a central r in several neurodegenerative disorders because of the presence of insoluble alpha-synuclein as the major fibrilla component of inclusion bodies. From the cloning of the human alpha-synuclein cDNA in 1993 to the present, alpl synuclein has been carefully documented in many aspects this article, we review the progress of studies on alpha-

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=> S Lewy body OR alpha-synuclein
  23 FILES SEARCHED...
  36 FILES SEARCHED...
  58 FILES SEARCHED...
  71 FILES SEARCHED...
L1       31933 LEWY BODY OR ALPHA-SYNUCLEIN

=> S L1 Abeta
MISSING OPERATOR L1 ABETA
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> S L1 AND abeta
  29 FILES SEARCHED...
  62 FILES SEARCHED...
L2        1623 L1 AND ABETA

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DRUGMONOG2, FEDRIP, FOREGE, GENBANK, IMSPRODUCT, IMSRESEARCH, KOSMET,
MEDICONF, NUTRACEUT, PCTGEN, PHAR, PHARMAML, PROUSDDR, PS, RDISCLOSURE,
SYNTHLINE'.
ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE
PROCESSING COMPLETED FOR L2
L3          1466 DUP REM L2 (157 DUPLICATES REMOVED)

=> S L3 AND immunization
  15 FILES SEARCHED...
  32 FILES SEARCHED...
  47 FILES SEARCHED...
  62 FILES SEARCHED...
L4            21 L3 AND IMMUNIZATION

=> D L4 1-21

L4   ANSWER 1 OF 21  BIOSIS  COPYRIGHT (c) 2005 The Thomson Corporation  on STN
AN   2004:355388  BIOSIS
DN   PREV200400362351
TI   Overcoming antigen masking of anti-amyloidbeta antibodies reveals breaking
     of B cell tolerance by virus-like particles in amyloidbeta immunized
     amyloid precursor protein transgenic mice.
AU   Li, Qingyou; Cao, Chuanhai; Chackerian, Bryce; Schiller, John; Gordon,
     Marcia; Ugen, Kenneth E.; Morgan, Dave [Reprint Author]
CS   Dept PharmacolAlzheimers Res Lab, Univ S Florida, Tampa, FL, 33612, USA
     qli@hsc.usf.edu; ccao@hsc.usf.edu; brycec@nih.gov;
     schillej@dc37a.nci.nih.gov; mgordon@hsc.usf.edu; kugen@hsc.usf.edu;
     dmorgan@hsc.usf.edu
SO   BMC Neuroscience, (June 8 2004) Vol. 5, No. June 8. print.
     ISSN: 1471-2202 (ISSN online).
DT   Article
LA   English
ED   Entered STN: 5 Sep 2004
     Last Updated on STN: 5 Sep 2004

L4    ANSWER 2 OF 21  BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN
AN    2002-13062  BIOTECHDS
TI    Composition comprising fusion protein which comprises binding portion
      that binds amyloid peptide epitope and a segment crossing blood-brain
      barrier, useful for treating disease states associated with amyloidosis;
         recombinant protein gene production, fusion protein composition,
         vector expression in host cell, useful in disease therapy and
         diagnosis
AU    NICOLAU Y C
PA    AVENTIS PHARMA SA; UNIV PASTEUR LOUIS
PI    WO 2002021141 14 Mar 2002
AI    WO 2000-US27632 6 Sep 2000
```

PRAI US 2000-255033 12 Dec 2000
DT Patent
LA English
OS WPI: 2002-351803 [38]

L4 ANSWER 3 OF 21 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2004:412731 CAPLUS
DN 140:417964
TI Prevention and treatment of synucleinopathic disease by administering agents that induce a beneficial immunogenic response against ***Lewy*** ***bodies***
IN Schenk, Dale B.; Masliah, Eliezer
PA Elan Pharmaceuticals, Inc., USA; The Regents of the University of California
SO PCT Int. Appl., 78 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 9

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004041067	A2	20040521	WO 2003-US34527	20031031

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2002-423012P P 20021101

L4 ANSWER 4 OF 21 IFIPAT COPYRIGHT 2005 IFI on STN
AN 10243450 IFIPAT;IFIUDB;IFICDB
TI NOVEL METHOD FOR DOWN-REGULATION OF AMYLOID; ADMINISTERING AN AMYLOIDOGENIC POLYPEPTIDE OR SUBSEQUENCE THEREOF TO INDUCES PRODUCTION OF ANTIBODIES AGAINST THE AMYLOIDOGENIC POLYPEPTIDE; TREATMENT OF ALZHEIMER'S DISEASE
IN Jensen Martin Roland (DK); Nielsen Klaus Gregorius (DK); Rasmussen Peter Birk (DK)
PA Unassigned Or Assigned To Individual (68000)
PI US 2002187157 A1 20021212
AI US 2001-785215 20010220
PRAI PA 2000-265 20000221
US 2000-186295P 20000301 (Provisional)
FI US 2002187157 20021212
DT Utility; Patent Application - First Publication **
00001000
FS CHEMICAL
APPLICATION
CLMN 58
GI 1 Figure(s).
FIG. 1: Schematic depiction of Autovac variants derived from the amyloid precursor protein with the purpose of generating antibody responses against the A beta protein A beta-43 (or C100). The APP is shown schematically at the top of the figure and the remaining schematic constructs show that the model epitopes P2 and P30 are substituted or inserted into various truncations of APP. In the figure, the black pattern indicates the APP signal sequence, two-way cross-hatching is the extracellular part of APP, dark vertical hatching is the transmembrane domain of APP, light vertical hatching is the intracellular domain of APP, coarse cross-hatching indicates the P30 epitope, and fine cross-hatching indicates the P2 epitope. The full line box indicates A beta-42/43 and the fullline box and the dotted line box together indicate C-100. " ***Abeta*** " denotes A beta .

L4 ANSWER 5 OF 21 USPATFULL on STN
AN 2005:36976 USPATFULL
TI Therapeutic formulations for the treatment of beta-amyloid related diseases

```
IN      Gervais, Francine, Ile Bizard, CANADA
        Bellini, Francesco, Mount Royal, CANADA
PI      US 2005031651       A1   20050210
AI      US 2004-871537      A1   20040618 (10)
RLI     Continuation-in-part of Ser. No. US 2003-746138, filed on 24 Dec 2003,
        PENDING
PRAI    WO 2003-CA2011           20031224
        US 2002-436379P          20021224 (60)
        US 2003-482214P          20030623 (60)
        US 2003-480984P          20030623 (60)
        US 2003-512116P          20031017 (60)
        US 2003-482058P          20030623 (60)
        US 2003-512135P          20031017 (60)
        US 2003-480918P          20030623 (60)
        US 2003-512017P          20031017 (60)
        US 2003-480906P          20030623 (60)
        US 2003-512047P          20031017 (60)
DT      Utility
FS      APPLICATION
LN.CNT 7983
INCL    INCLM: 424/400.000
NCL     NCLM:  424/400.000
IC      [7]
        ICM: A61K009-00
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4    ANSWER 6 OF 21  USPATFULL on STN
AN      2005:10897  USPATFULL
TI      Genes and polymorphisms on chromosome 10 associated with Alzheimer's
        disease and other neurodegenerative diseases
IN      Becker, Kenneth David, San Diego, CA, UNITED STATES
        Velicelebi, Gonul, San Diego, CA, UNITED STATES
        Elliott, Kathryn J., San Diego, CA, UNITED STATES
        Wang, Xin, San Diego, CA, UNITED STATES
        Tanzi, Rudolph E., Hull, MA, UNITED STATES
        Bertram, Lars, Boston, MA, UNITED STATES
        Saunders, Aleister J., Philadelphia, PA, UNITED STATES
        Mullin, Kristina M., Weymouth, MA, UNITED STATES
        Sampson, Andrew Joseph, Oakwood, OH, UNITED STATES
PI      US 2005009031       A1   20050113
AI      US 2003-600009      A1   20030618 (10)
RLI     Continuation-in-part of Ser. No. US 2002-282174, filed on 25 Oct 2002,
        PENDING Continuation-in-part of Ser. No. WO 2002-US34679, filed on 25
        Oct 2002, PENDING
PRAI    US 2001-339525P          20011025 (60)
        US 2001-338010P          20011108 (60)
        US 2001-336929P          20011108 (60)
        US 2001-338363P          20011109 (60)
        US 2001-337052P          20011204 (60)
        US 2002-368919P          20020328 (60)
DT      Utility
FS      APPLICATION
LN.CNT 15528
INCL    INCLM: 435/006.000
        INCLS: 536/024.300
NCL     NCLM:  435/006.000
        NCLS:  536/024.300
IC      [7]
        ICM: C12Q001-68
        ICS: C07H021-04
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4    ANSWER 7 OF 21  USPATFULL on STN
AN      2004:334837  USPATFULL
TI      Method for the prediction, diagnosis and differential diagnosis of
        Alzheimer's disease
IN      Vanderstichele, Hugo, Gent, BELGIUM
        Vanmechelen, Eugeen, Nażareth-Eke, BELGIUM
        De Meyer, Geert, Gent, BELGIUM
        Blennow, Kaj, Goteborg, SWEDEN
        Kostanjevecki, Vesna, Sint-Denijs-Westrem, BELGIUM
PI      US 2004265919       A1   20041230
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AI      US 2004-848686      A1   20040519 (10)
PRAI    EP 2003-447120           20030522
        US 2003-477621P          20030611 (60)
DT      Utility
FS      APPLICATION
LN.CNT 2860
INCL    INCLM: 435/007.200
NCL     NCLM:  435/007.200
IC      [7]
        ICM: G01N033-53
        ICS: G01N033-567
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4    ANSWER 8 OF 21  USPATFULL on STN
AN      2004:327335  USPATFULL
TI      Methods of engineering spatially conserved motifs in polypeptides
IN      Chan, John, Raleigh, NC, UNITED STATES
        Zhang, Shengsheng, Framingham, MA, UNITED STATES
        Baynes, Brian, Somerville, MA, UNITED STATES
PA      Compound Therapeutics, Inc., Waltham, MA (U.S. corporation)
PI      US 2004259155       A1   20041223
AI      US 2003-676873      A1   20030930 (10)
PRAI    US 2002-414688P          20020930 (60)
DT      Utility
FS      APPLICATION
LN.CNT 2745
INCL    INCLM: 435/007.100
        INCLS: 702/019.000
NCL     NCLM:  435/007.100
        NCLS:  702/019.000
IC      [7]
        ICM: G01N033-53
        ICS: G06F019-00; G01N033-48; G01N033-50
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4    ANSWER 9 OF 21  USPATFULL on STN
AN      2004:120535  USPATFULL
TI      Peptides and methods of screening immunogenic peptide vaccines against
        Alzheimer's Disease
IN      Fitzer-Attas, Cheryl, Rehovot, ISRAEL
        Chain, Daniel G., Jerusalem, ISRAEL
PI      US 2004091945       A1   20040513
AI      US 2003-619454      A1   20030716 (10)
PRAI    US 2002-396245P          20020717 (60)
DT      Utility
FS      APPLICATION
LN.CNT 2604
INCL    INCLM: 435/007.200
        INCLS: 530/350.000
NCL     NCLM:  435/007.200
        NCLS:  530/350.000
IC      [7]
        ICM: G01N033-53
        ICS: G01N033-567; C07K014-47
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4    ANSWER 10 OF 21  USPATFULL on STN
AN      2004:107249  USPATFULL
TI      Adzymes and uses thereof
IN      Afeyan, Noubar B., Lexington, MA, UNITED STATES
        Lee, Frank D., Chestnut Hill, MA, UNITED STATES
        Wong, Gordon G., Brookline, MA, UNITED STATES
        Das Gupta, Ruchira, Auburndale, MA, UNITED STATES
        Baynes, Brian, Somerville, MA, UNITED STATES
PI      US 2004081648       A1   20040429
AI      US 2003-650592      A1   20030827 (10)
PRAI    US 2002-406517P          20020827 (60)
        US 2002-423754P          20021105 (60)
        US 2002-430001P          20021127 (60)
DT      Utility
FS      APPLICATION
LN.CNT 8325
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INCL    INCLM: 424/094.630
        INCLS: 435/226.000
NCL     NCLM:  424/094.630
        NCLS:  435/226.000
IC      [7]
        ICM: A61K038-48
        ICS: C12N009-64
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4    ANSWER 11 OF 21  USPATFULL on STN
AN      2004:107248  USPATFULL
TI      Adzymes and uses thereof
IN      Afeyan, Noubar B., Lexington, MA, UNITED STATES
        Lee, Frank D., Chestnut Hill, MA, UNITED STATES
        Wong, Gordon G., Brookline, MA, UNITED STATES
        DasGupta, Ruchira, Auburndale, MA, UNITED STATES
        Baynes, Brian, Somerville, MA, UNITED STATES
PI      US 2004081647       A1    20040429
AI      US 2003-650591      A1    20030827 (10)
PRAI    US 2002-406517P      20020827 (60)
        US 2002-423754P      20021105 (60)
        US 2002-430001P      20021127 (60)
DT      Utility
FS      APPLICATION
LN.CNT 7919
INCL    INCLM: 424/094.630
        INCLS: 435/069.700; 435/226.000
NCL     NCLM:  424/094.630
        NCLS:  435/069.700; 435/226.000
IC      [7]
        ICM: A61K038-48
        ICS: C12N009-64; C12P021-04
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4    ANSWER 12 OF 21  USPATFULL on STN
AN      2004:51633  USPATFULL
TI      Amine 1,2- and 1,3-diol compounds
IN      Romero, Arthur G., Kalamazoo, MI, UNITED STATES
        Schostarez, Heinrich J., Portage, MI, UNITED STATES
        Roels, Christina M., Battle Creek, MI, UNITED STATES
PI      US 2004039064       A1    20040226
AI      US 2002-299739      A1    20021119 (10)
PRAI    US 2001-333081P      20011119 (60)
        US 2001-334000P      20011128 (60)
        US 2002-362752P      20020308 (60)
DT      Utility
FS      APPLICATION
LN.CNT 10130
INCL    INCLM: 514/651.000
        INCLS: 564/355.000
NCL     NCLM:  514/651.000
        NCLS:  564/355.000
IC      [7]
        ICM: A61K031-137
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4    ANSWER 13 OF 21  USPATFULL on STN
AN      2004:44501  USPATFULL
TI      Proteins and nucleic acids encoding same
IN      Tchernev, Velizar T., Branford, CT, UNITED STATES
        Spytek, Kimberly A., New Haven, CT, UNITED STATES
        Zerhusen, Bryan D., Branford, CT, UNITED STATES
        Patturajan, Meera, Branford, CT, UNITED STATES
        Shimkets, Richard A., West Haven, CT, UNITED STATES
        Li, Li, Branford, CT, UNITED STATES
        Gangolli, Esha A., Madison, CT, UNITED STATES
        Padigaru, Muralidhara, Branford, CT, UNITED STATES
        Anderson, David W., Branford, CT, UNITED STATES
        Rastelli, Luca, Guilford, CT, UNITED STATES
        Miller, Charles E., Hill Drive, CT, UNITED STATES
        Gerlach, Valerie, Branford, CT, UNITED STATES
        Taupier, Raymond J., JR., East Haven, CT, UNITED STATES
```

Gusev, Vladimir Y., UNITED STATES
Colman, Steven D., Guilford, CT, UNITED STATES
Wolenc, Adam Ryan, New Haven, CT, UNITED STATES
Pena, Carol E. A., Guilford, CT, UNITED STATES
Furtak, Katarzyna, Anosia, CT, UNITED STATES
Grosse, William M., Bransford, CT, UNITED STATES
Alsobrook, John P., II, Madison, CT, UNITED STATES
Lepley, Denise M., Branford, CT, UNITED STATES
Rieger, Daniel K., Branford, CT, UNITED STATES
Burgess, Catherine E., Wethersfield, CT, UNITED STATES

PI	US 2004033493	A1	20040219	
AI	US 2002-72012	A1	20020131	(10)
PRAI	US 2001-267459P		20010208	(60)
	US 2001-266975P		20010207	(60)
	US 2001-267057P		20010207	(60)
	US 2001-266767P		20010205	(60)
	US 2001-266406P		20010202	(60)
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	US 2001-265514P		20010131	(60)
	US 2001-267823P		20010209	(60)
	US 2001-268974P		20010215	(60)
	US 2001-271855P		20010227	(60)
	US 2001-271839P		20010227	(60)
	US 2001-273046P		20010302	(60)
	US 2001-272788P		20010302	(60)
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	US 2001-275925P		20010314	(60)
	US 2001-275947P		20010314	(60)
	US 2001-275950P		20010314	(60)
	US 2001-276450P		20010315	(60)
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	US 2001-278652P		20010320	(60)
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	US 2001-278778P		20010326	(60)
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	US 2001-283083P		20010411	(60)
	US 2001-282992P		20010411	(60)
	US 2001-285133P		20010420	(60)
	US 2001-285749P		20010423	(60)
	US 2001-288327P		20010503	(60)
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	US 2001-323379P		20010919	(60)
	US 2001-330308P		20011018	(60)
	US 2001-330245P		20011018	(60)
	US 2001-332701P		20011114	(60)
	US 2001-271664P		20010226	(60)

DT Utility
FS APPLICATION
LN.CNT 59681
INCL INCLM: 435/006.000
INCLS: 435/007.230; 435/069.300; 435/320.100; 435/325.000; 530/350.000; 536/023.200; 435/183.000; 424/155.100

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NCL     NCLM:  435/006.000
        NCLS:  435/007.230; 435/069.300; 435/320.100; 435/325.000; 530/350.000;
               536/023.200; 435/183.000; 424/155.100
IC      [7]
        ICM: C12Q001-68
        ICS: G01N033-574; C07H021-04; A61K039-395; C12N009-00; C12P021-02;
        C12N005-06; C07K014-47
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4    ANSWER 14 OF 21  USPATFULL on STN
AN      2004:18378  USPATFULL
TI      Neurotoxic oligomers
IN      Bush, Ashley, Somerville, MA, UNITED STATES
        Cherny, Robert, Victoria, AUSTRALIA
PI      US 2004013680       A1    20040122
AI      US 2003-312437      A1    20030616 (10)
        WO 2001-AU786             20010628
DT      Utility
FS      APPLICATION
LN.CNT 1214
INCL    INCLM: 424/185.100
        INCLS: 530/400.000
NCL     NCLM:  424/185.100
        NCLS:  530/400.000
IC      [7]
        ICM: A61K039-00
        ICS: C07K014-47
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4    ANSWER 15 OF 21  USPATFULL on STN
AN      2003:325156  USPATFULL
TI      Aza hydroxylated ethyl amine compounds utility
IN      Schostarez, Heinrich, Portage, MI, UNITED STATES
        Chrusciel, Robert Alan, Portage, MI, UNITED STATES
        Centko, Rebecca S., Portage, MI, UNITED STATES
PI      US 2003229138       A1    20031211
AI      US 2002-152601      A1    20020522 (10)
PRAI    US 2001-292856P      20010522 (60)
        US 2001-301355P      20010627 (60)
        US 2002-360601P      20020301 (60)
DT      Utility
FS      APPLICATION
LN.CNT 6845
INCL    INCLM: 514/526.000
        INCLS: 514/664.000; 514/561.000; 514/651.000; 514/614.000; 558/446.000;
               562/561.000; 564/147.000; 564/347.000; 564/464.000; 558/445.000
NCL     NCLM:  514/526.000
        NCLS:  514/664.000; 514/561.000; 514/651.000; 514/614.000; 558/446.000;
               562/561.000; 564/147.000; 564/347.000; 564/464.000; 558/445.000
IC      [7]
        ICM: A61K031-275
        ICS: A61K031-195; A61K031-16; A61K031-15; A61K031-137
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4    ANSWER 16 OF 21  USPATFULL on STN
AN      2003:318636  USPATFULL
TI      Genes and polymorphisms on chromosome 10 associated with Alzheimer's
        disease and other neurodegenerative diseases
IN      Becker, Kenneth David, San Diego, CA, UNITED STATES
        Velicelebi, Gonul, San Diego, CA, UNITED STATES
        Ellliott, Kathryn J., San Diego, CA, UNITED STATES
        Wang, Xin, San Diego, CA, UNITED STATES
        Tanzi, Rudolph E., Hull, MA, UNITED STATES
        Bertram, Lars, Brighton, MA, UNITED STATES
        Saunders, Aleister J., Philadelphia, PA, UNITED STATES
        Mullin, Kristina M., south Boston, MA, UNITED STATES
        Sampson, Andrew Joseph, Dayton, OH, UNITED STATES
PA      The General Hospital Corporation (U.S. corporation)
PI      US 2003224380       A1    20031204
AI      US 2002-282174      A1    20021025 (10)
PRAI    US 2001-339525P      20011025 (60)
        US 2001-338010P      20011108 (60)
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       US 2001-336929P     20011108 (60)
       US 2001-338363P     20011109 (60)
       US 2001-337052P     20011204 (60)
       US 2002-368919P     20020328 (60)
       US 2001-348065P     20011025 (60)
       US 2001-336983P     20011102 (60)
DT     Utility
FS     APPLICATION
LN.CNT 13662
INCL   INCLM: 435/006.000
NCL    NCLM:  435/006.000
IC     [7]
       ICM: C12Q001-68
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4   ANSWER 17 OF 21  USPATFULL on STN
AN     2003:225306  USPATFULL
TI     Novel method for down-regulation of amyloid
IN     Rasmussen, Peter Birk, Horsholm, DENMARK
       Jensen, Martin Roland, Horsholm, DENMARK
       Nielsen, Klaus Gregorius, Horsholm, DENMARK
       Koefoed, Peter, Horsholm, DENMARK
       Degan, Florence Dal, Horsholm, DENMARK
PI     US 2003157117      A1   20030821
AI     US 2002-223809     A1   20020820 (10)
PRAI   DK 2001-1231        20010820
       DK 2002-58          20020416
       US 2001-337543P     20011022 (60)
       US 2002-373027P     20020416 (60)
DT     Utility
FS     APPLICATION
LN.CNT 3681
INCL   INCLM: 424/185.100
       INCLS: 435/226.000
NCL    NCLM:  424/185.100
       NCLS:  435/226.000
IC     [7]
       ICM: A61K039-00
       ICS: C12N009-64
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4   ANSWER 18 OF 21  USPATFULL on STN
AN     2003:135733  USPATFULL
TI     Transgenic animal model of neurodegenerative disorders
IN     St. George-Hyslop, Peter H., Toronto, CANADA
       Fraser, Paul E., Toronto, CANADA
       Westaway, David, Etobicoke, CANADA
PI     US 2003093822      A1   20030515
AI     US 2001-884629     A1   20010619 (9)
PRAI   US 2000-212534P     20000620 (60)
DT     Utility
FS     APPLICATION
LN.CNT 1380
INCL   INCLM: 800/018.000
       INCLS: 800/012.000
NCL    NCLM:  800/018.000
       NCLS:  800/012.000
IC     [7]
       ICM: A01K067-027

L4   ANSWER 19 OF 21  USPATFULL on STN
AN     2003:126727  USPATFULL
TI     Novel methods for down-regulation of amyloid
IN     Jensen, Martin Roland, Horsholm, DENMARK
       Birk, Peter, Horsholm, DENMARK
       Nielsen, Klaus Gregorius, Horsholm, DENMARK
PI     US 2003086938      A1   20030508
AI     US 2002-204362     A1   20020816 (10)
       WO 2001-DK113           20010219
PRAI   DK 2000-265         20000221
DT     Utility
FS     APPLICATION
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LN.CNT 3114
INCL    INCLM: 424/185.100
NCL     NCLM:  424/185.100
IC      [7]
        ICM: A61K039-00
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4    ANSWER 20 OF 21  USPATFULL on STN
AN      2002:191196  USPATFULL
TI      Methods and compositions for stimulating CD 45 and  thereby suppressing
        microglial activation  associated with Alzheimer's disease
IN      Tan, Jun, Tampa, FL, UNITED STATES
        Town, Terrence, Tampa, FL, UNITED STATES
        Mullan, Michael, Tampa, FL, UNITED STATES
PI      US 2002102259       A1    20020801
AI      US 2001-985598      A1    20011105 (9)
PRAI    US 2000-245608P      20001103 (60)
DT      Utility
FS      APPLICATION
LN.CNT 1744
INCL    INCLM: 424/144.100
NCL     NCLM:  424/144.100
IC      [7]
        ICM: A61K039-395
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4    ANSWER 21 OF 21  WPIDS  COPYRIGHT 2005 THE THOMSON CORP on STN
AN    2002-130835 [17]   WPIDS
DNC   C2002-040215
TI    Treatment and prevention of conditions characterized by pathological
      aggregation and accumulation of a specific protein associated with
      oxidative damage and tyrosine-cross-link formation..
DC    B04 D16
IN    BUSH, A; CHERNY, R
PA    (GEHO) GEN HOSPITAL CORP; (PRAN-N) PRANA BIOTECHNOLOGY LTD; (BUSH-I) BUSH
      A; (CHER-I) CHERNY R
CYC   97
PI    WO 2002000245    A1 20020103 (200217)* EN    59    A61K038-16
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             NL OA PT SD SE SL SZ TR TZ UG ZW
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             DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR
             KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU
             SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
      AU 2001068828    A   20020108 (200235)             A61K038-16
      EP 1296705       A1 20030402 (200325)  EN          A61K038-16
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      CN 1450908       A   20031022 (200406)             A61K038-16
      US 2004013680    A1 20040122 (200407)             A61K039-00
      JP 2004501204    W   20040115 (200410)       96    A61K038-00
ADT   WO 2002000245 A1 WO 2001-AU786 20010628; AU 2001068828 A AU 2001-68828
      20010628; EP 1296705 A1 EP 2001-947033 20010628, WO 2001-AU786 20010628;
      CN 1450908 A CN 2001-813312 20010628; US 2004013680 A1 WO 2001-AU786
      20010628, US 2003-312437 20030616; JP 2004501204 W WO 2001-AU786 20010628,
      JP 2002-505026 20010628
FDT   AU 2001068828 A Based on WO 2002000245; EP 1296705 A1 Based on WO
      2002000245; JP 2004501204 W Based on WO 2002000245
PRAI  US 2000-242177P      20001023; US 2000-214779P      20000628;
      US 2003-312437       20030616
IC    ICM  A61K038-00; A61K038-16; A61K039-00
      ICS  A61K039-395; A61P021-00; A61P025-02; A61P025-14; A61P025-16;
           A61P025-28; A61P027-12; C07K014-47; G01N033-53
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